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LETTER TO THE EDITOR

Vitamin B_{12} deficiency, seizure, involuntary movements

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I read with interest the article by Benbir et al.[1] The paper of the authors seems to change the knowledge about seizure and Vitamin B_{12} deficiency (VBD). In that paper, three infants with VBD were reported because of seizures following the initiation of intramuscular Vitamin B_{12} treatment. VBD may cause the involuntary movements (IM) and seizures. The IM can be commonly confused with seizure. I think to look over the IM and seizures.

The IM are one of the impressive characteristics on VBD. Some of the patients can exhibit the IM as choreoathetoid movements,[2] rolling movements,[3] jerky movements,[4] tremor[5] before treatment. However some IM such as shaking movements,[6] myoclonus,[7,8] tremor and chorea,[9,10] twitching,[10] protrusion or tremor of the tongue,[9,10] wandering eye movements[11] become visible after the beginning of the treatment. In these cases the IM may appear 2 days after the initiation of cobalamin therapy,[6] usually continue 2—3 weeks.[6,9] It may be observed at the third month of treatment.[10] Katar et al.[5] reported prevalence of tremor as 12% before treatment. These movements may persist in sleeping[6] or disappear during sleeping.[9]

The reports about epileptic seizures of VBD are not much. Generalize tonic-clonic seizure,[12,13] focal seizure,[14] infantile spasm[15] were described as VBD related seizures. The seizures in these cases usually begin before the treatment with Vitamin B_{12}. The therapy can stop the seizures, promptly.[12,13] Some patients exhibit therapy resistant seizures.[14,16] In the case of Korenke et al.,[13] a tremor as the first neurological symptom was observed, then manifested grand-mal seizures. The seizures which occurred after Vitamin B_{12} therapy are fairly few and appeared later period. Johnson and Roloff[17] reported a patient who began having myoclonic seizures, confirmed by EEG 3 months after discharge. Additionally, two children had IM at the diagnosis (myoclonic spasms and choreiform movements), continued to have poorly controlled seizures.[16] Two of four cases who had seizures before Vitamin B_{12} administration were associated long-term sequelae like retardation.[12—15] All the cases with seizure, which appeared after vitamin therapy, were also retarded.[16,17] So nutritional VBD related seizures may not be benign. If VBD belongs to inborn error of cobalamin metabolism, the prognosis may be worse.[18]

The EEG of patients with VBD and seizure may show various features including hypsarrhythmia.[14,15] Lundgren and Blennow[12] found seizure activity at fourth EEG of their patient with no clinical symptoms who had generalized tonic-clonic convulsions before. The case reported by Korenko et al.[13] had an interesting EEG course. Generalized slow activity was seen on the EEG of the female infant with generalized tonic-clonic seizure at the diagnosis. Parenteral cobalamin stopped convulsions within 24 h. Although no further convulsions, an EEG showed epileptic discharges at the fourth month of therapy. Further EEGs were normal. The EEG of the case reported by Johnson and Roloff[17] was consistent with a seizure disorder of multifocal cortical origin and diffuse cortical dysfunction at first. A repeat EEG, 3 weeks after initiation of therapy, was normal. Approximately, 3 months later, he began having myoclonic seizures confirmed by EEG.

The EEG of patients with IM, before therapy, may demonstrate slowing of the basal activity compatible with diffuse encephalopathy.[2,3] The patients who exhibited IM after vitamin treatment may have a normal EEG before and after therapy,[6] slow background activity before therapy and a normal EEG after therapy.[9] Stollhoff and Schulte[7] reported a case with different EEG course. The first EEG showed

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general slowing and multifocal sharp waves. In the first week, after initiation of vitamin therapy, rhythmic discharges of sharp waves and spike activity and continuous myoclonic jerks were encountered. The EEG normalized after the 5 weeks of therapy. In my opinion, the EEG may not be sufficient in the discrimination between the seizure and the IM. The use of video-EEG monitoring may be profitable in complex cases.

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